

_____)
G.B., widow of R.B., Appellant)
)
 and)
)
)
DEPARTMENT OF THE NAVY, LONG)
BEACH NAVAL SHIPYARD, Long Beach, CA,)
Employer)
 _____)
)

Docket No. 16-1363
Issued: March 2, 2017

Case Submitted on the Record

Before:
CHRISTOPHER J. GODFREY, Chief Judge
PATRICIA H. FITZGERALD, Deputy Chief Judge
ALEC J. KOROMILAS, Alternate Judge

² 5 U.S.C. § 8101 *et seq.*

ISSUE

The issue is whether the employee's death on September 7, 2011 was causally related to factors of his federal employment.

FACTUAL HISTORY

The employee, then a 43-year-old pipefitter and insulator, filed an occupational disease claim (Form CA-2) on September 24, 1979, alleging that he developed asbestosis causally related to factors of his federal employment. OWCP accepted a claim for asbestosis and the employee stopped work in 1980. It commenced payment for total disability and received compensation on the periodic rolls from December 2, 1981 until the date of his death on September 7, 2011.

The September 15, 2011 certificate of death listed the causes of death as advanced pulmonary fibrosis due to asbestosis, chronic obstructive lung disease, and ischemic heart disease.

On September 17, 2011 appellant filed a claim for death benefits (Form CA-5), alleging that her husband's death was causally related to his accepted asbestosis conditions.

In a September 21, 2011 attending physician's form report, Dr. Mukesh M. Patel, Board-certified in cardiology and internal medicine and the employee's attending physician, opined that the direct cause of death was pulmonary fibrosis and that the employee's death was due to the accepted work-related injury because of asbestosis at work. He asserted that the employee developed advanced pulmonary insufficiency due to pulmonary fibrosis.

In a hospital admission report dated August 15, 2011, received by OWCP on January 3, 2012, Dr. Patel advised that the employee had been experiencing shortness of breath and dyspnea and had a massive edema of the leg. He noted that the employee had congestive heart failure, with underlying right and left heart failure, biventricular failure, systolic and diastolic dysfunction of the left ventricle, an underlying history of implantable cardioverter defibrillator, end-stage lung disease, and pulmonary fibrosis.

In a hospital report/discharge summary dated September 5, 2011, received by OWCP on October 21, 2011, Dr. Patel advised that the employee had been admitted because of shortness of breath, with underlying right and left heart failure, severe pulmonary fibrosis, and pneumonia. The employee was released and sent home for hospice care. Dr. Patel reported that the employee also had end-stage chronic obstructive pulmonary disease and ischemic cardiomyopathy.

On March 12, 2012 OWCP received a report from Dr. Edward C. Federman, Board-certified in internal medicine and pulmonary disease, and an OWCP medical adviser. Dr. Federman opined that the question of whether the employee's death was caused by asbestos-related lung disease was complex. He noted that initial studies of asbestos were suggestive that the inflammatory and thrombogenic effects of pulmonary exposure to asbestos might increase the risk of cardiovascular disease. Dr. Federman advised, however, that further epidemiologic studies of asbestos workers did not support atherosclerosis being provoked by asbestos exposure.

He noted that there was an increased risk of respiratory disease with pneumoconiosis, pleural cancer, and lung cancer being increased, as well as peritoneal cancer in the subgroup of asbestos workers, but no indication of increased risk of atherosclerotic cardiovascular disease or congestive heart failure within this group.

Dr. Federman concluded that the employee apparently had advanced atherosclerosis, congestive heart failure, and arrhythmias, which were the precipitating cause of his death, exacerbated by type 2 diabetes mellitus and cigarette exposure, as well as asbestos exposure. He opined that there was no evidence of acceleration of his atherosclerosis from the occupational asbestos exposure.

In a decision dated May 18, 2012, OWCP denied appellant's claim, finding that the evidence of record failed to establish that the employee's death was due to factors of his federal employment.

On February 8, 2013 appellant, through counsel, requested reconsideration of the May 18, 2012 decision.

In a March 19, 2013 report, Dr. Patel reiterated his opinion that the employee's death was due at least in part to his work-related asbestosis condition. He advised that the problem of asbestosis was very common in ship builders. Dr. Patel noted that the employee was exposed to asbestos for almost 13 years and underwent x-rays, which revealed classical bilateral pulmonary fibrosis. He reported that the employee inhaled asbestos fibers, which was the reason he started to have continuous slow progression of the disease over the years, and then became totally disabled. Dr. Patel noted that he had reviewed the employee's chest x-rays throughout this period, all of which revealed significant bilateral pulmonary fibrosis. He also advised that the employee developed restrictive lung disease, impaired diffusing capacity, and right heart failure. Because the employee had right heart failure over a long period of time, he ultimately went into left heart failure, and he was dependent on oxygen. Dr. Patel reported that the onset was insidious and that the employee had progressive dyspnea over the years, with a dry cough, a decrease in exercise tolerance, expiratory crackles, and wheezing, with forced exhalation and markedly reduced lung capacity. He opined:

"In my opinion, (the employee) became totally disabled because he was exposed to asbestos during working at the [shipyard], which led him to his detrimental condition all along and precipitated all his other problems. Because of his asbestosis, he had marked right heart failure and subsequently he needed an implantable automatic cardioverter defibrillator which I placed, and he started to have ventricular tachycardia. [The employee] also started to have swelling of the lower extremities. He was extremely dyspneic throughout. [The employee's] terminal event was that he died because of asbestosis-related problems, like right heart failure ultimately leading to left heart failure, ultimately leading to extremely decreased oxygen diffusion capacity, which took away his life."

By decision dated July 25, 2013, OWCP denied modification of the May 18, 2012 decision. It found that the weight of medical evidence remained with Dr. Federman's report, which was well reasoned and supported by relevant medical literature. OWCP further noted that

Dr. Federman is a specialist in the type of condition being claimed as causing the employee's death.

On November 11, 2013 appellant, through counsel, requested reconsideration of the July 25, 2013 decision.

In a January 27, 2014 report, Dr. Federman advised that the employee had a 70 to 80 pack a year smoking history and quit smoking in 1979. He noted that the employee had a myocardial infarction in 1979 and had cardiovascular problems for the remaining years of his life; coronary artery bypass surgery was performed in January 2001. Dr. Federman reported that the employee also developed type 2 diabetes mellitus at that time and had problems with arrhythmias. The employee required a pacemaker placement, and had a cardioversion for atrial fibrillation ventricular tachycardia in February 2006. Dr. Federman noted that the employee was admitted to the hospital on September 5, 2011 with chronic kidney disease, as well as atherosclerotic cardiovascular disease, and congestive heart failure and was placed on hospice care at that time.

Dr. Federman advised that he had reviewed a medical study which showed that former smokers had an elevated risk of ischemic heart disease. He inferred from this study that asbestos is an independent contributor to cardiovascular disease, presumably from its inflammatory effect, but that smoking is a much more profound cause of inflammation. Dr. Federman explained, given that the addition of asbestos to smoking did not increase the risk, this implied that smoking causes maximal inflammation and asbestos does not add to the process. He opined that, given that the employee was still smoking around the time of his heart attack, this would indicate no additional inflammation from his asbestos exposure, at least as it related to his atherosclerotic heart disease. Dr. Federman advised that his initial reports did not support the correlation between the asbestos exposure and atherosclerosis. He asserted that the possibility of some acceleration on the basis of asbestos exposure seemed unlikely given the new epidemiologic studies presented. Dr. Federman opined that this additional information reinforced and confirmed his initial evaluation that there was no contributing cause from the employee's exposure to asbestos to his cardiovascular disease. He therefore reiterated his opinion that the cause of the atherosclerotic cardiovascular disease that the employee had was due to his cigarette smoking history and type 2 diabetes mellitus.

By decision dated February 12, 2014, OWCP denied modification of its prior decision. It found that Dr. Federman's opinion continued to represent the weight of medical opinion evidence in the case, noting that his January 27, 2014 report again provided medical rationale explaining why the employee's death was not be related to his exposure to asbestos.

On November 10, 2014 appellant, through counsel, requested reconsideration.

In an August 21, 2014 report, received by OWCP on November 10, 2014, Dr. Patel advised that the employee was directly exposed to asbestos and he opined that patients with asbestosis were subject to a high risk of related lung disease through inhalation of the fibers. He noted that during the employee's years of exposure to asbestos, workers were not provided with masks. Dr. Patel reported that the employee had a deteriorating lung condition, exacerbation of chronic obstructive pulmonary disease, and pneumonitis. He opined that all of these problems

were due to the employee's underlying asbestos exposure. Dr. Patel further noted that the employee stopped smoking more than 30 years ago and that the employee also subsequently developed coronary artery disease and that he underwent open heart surgery. He advised that the employee's left heart failure and subsequent asbestosis both contributed to his death.

Dr. Patel noted that the employee underwent a computerized axial tomography scan of the chest in July 2011, suggestive of asbestosis, which created bilateral pneumonia, and right heart failure. He opined that his general condition gradually and markedly deteriorated and that ultimately, asbestosis took the employee's life. Dr. Patel concluded that he was very confident, without any doubt, that the cause of the employee's death was asbestosis.

In a report dated December 29, 2014, Dr. Federman advised that he reviewed Dr. Patel's August 21, 2014 report and additional records. He noted that there was no question that asbestos exposure increased inflammatory biomarkers and that, with animal models, there was evidence of increased progression of atherosclerotic cardiovascular disease. Dr. Federman reiterated, however, that the employee's atherosclerosis was caused by his long-term smoking habit, and was not causally related to his work-related asbestosis condition either by direct cause, aggravation, precipitation, or acceleration.

By decision dated January 26, 2015, OWCP denied modification of its February 12, 2014 decision.

On December 3, 2015 counsel requested reconsideration.

In an October 30, 2015 report, received by OWCP on December 3, 2015, Dr. Patel essentially reiterated his previous findings and conclusions.

By decision dated March 1, 2016, OWCP denied modification of its prior decision. It found that the weight of the medical evidence was represented by Dr. Federman's opinion that the employee's death was not causally related to a work-related condition. OWCP further found that Dr. Patel's additional reports did not support a finding that the employee's death was caused, precipitated, or accelerated by his asbestosis.

LEGAL PRECEDENT

Appellant has the burden of proving by the weight of the reliable, probative, and substantial evidence that the employee's death was causally related to his or her federal employment. This burden includes the necessity of furnishing medical opinion evidence of a cause and effect relationship based on a proper factual and medical background.³

The medical evidence required to establish causal relationship, generally, is rationalized medical opinion evidence.⁴ Rationalized medical opinion evidence is medical evidence which includes a physician's rationalized opinion on the issue of whether there is a causal relationship

³ *Kathy Marshall (James Marshall)*, 45 ECAB 827, 832 (1994); *Timothy Forsyth (James Forsyth)*, 41 ECAB 467, 470 (1990).

⁴ *See Naomi A. Lilly*, 10 ECAB 560, 572-73 (1959).

between the claimant's diagnosed condition and the implicated employment factors. The opinion of the physician must be based on a complete factual and medical background of the claimant,⁵ must be one of reasonable medical certainty,⁶ and must be supported by medical rationale explaining the nature of the relationship between the diagnosed condition and the specific employment factors identified by the claimant.⁷

It is not necessary to provide a significant contribution of employment factors for the purpose of establishing causal relationship.⁸

Section 8123(a) provides that, if there is a disagreement between the physician making the examination for the United States and the physician of the employee, the Secretary shall appoint a third physician who shall make an examination.⁹ It is well-established that, when a case is referred to an impartial medical specialist for the purpose of resolving a conflict, the opinion of such specialist, if sufficiently well rationalized and based on a proper factual and medical background, must be given special weight.¹⁰

ANALYSIS

Appellant filed a claim for compensation based on the September 7, 2011 death of the employee. The employee's death certificate listed the causes of death as advanced pulmonary fibrosis due to asbestosis, chronic obstructive lung disease, and ischemic heart disease. At the time of his death, the employee had an accepted claim for asbestosis and was receiving disability compensation. Appellant alleged that his accepted asbestosis caused or contributed to his advanced pulmonary fibrosis, chronic obstructive lung disease, and ischemic heart disease. The issue of causal relationship is a medical question.

Dr. Patel noted in the September 21, 2011 attending physician's form report that the direct cause of death was pulmonary fibrosis and that the employee's death was due to the accepted work-related injury. He noted that the employee developed advanced pulmonary insufficiency due to pulmonary fibrosis. In a hospital report/discharge summary dated September 5, 2011, received by OWCP on October 21, 2011, Dr. Patel advised that the employee had been admitted because of underlying short of breath, with underlying right and left heart failure, severe pulmonary fibrosis, and pneumonia. The employee was released to home for underlying hospice care. Dr. Patel reported that the employee also had end-stage chronic obstructive pulmonary disease and ischemic cardiomyopathy. In his March 19, 2013 and August 21, 2014 reports, Dr. Patel further opined that the employee's death was due at least in part to his work-related asbestosis condition. Dr. Patel advised that the employee was exposed to

⁵ *William Nimitz, Jr.*, 30 ECAB 567, 570 (1979).

⁶ *See Morris Scanlon*, 11 ECAB 384-85 (1960).

⁷ *See William E. Enright*, 31 ECAB 426, 430 (1980).

⁸ *See Richard E. Simpson*, 55 ECAB 490 (2004).

⁹ *Regina T. Pellicchia*, 53 ECAB 155 (2001).

¹⁰ *Jacqueline Brasch (Ronald Brasch)*, 52 ECAB 252 (2001).

asbestos for nearly almost 13 years and underwent x-rays, which revealed classical bilateral pulmonary fibrosis. He reported that the employee inhaled asbestos fibers, which was the reason he started to have continuous slow progression of the disease over the years, and then became totally disabled. Dr. Patel advised that he had reviewed the employee's chest x-rays throughout this period, all of which revealed significant bilateral pulmonary fibrosis. He opined that the employee became totally disabled because he was exposed to asbestos during work at the employing establishment, which led to his detrimental condition, and precipitated all his other problems.

Dr. Patel opined that, because of his asbestosis, the employee had marked right heart failure and subsequently needed an implantable automatic cardioverter defibrillator; the employee began to have ventricular tachycardia and died because of asbestosis-related problems, like right heart failure ultimately leading to left heart failure, ultimately leading to extremely decreased oxygen diffusion capacity. He concluded that he was very confident, without any doubt, that the cause of the employee's death was asbestosis.

Dr. Federman found in his January 27, 2017 report that there was no causal relationship between the employee's death and his accepted asbestosis. He noted that initial studies of asbestos were suggestive that the inflammatory and thrombogenic effects of pulmonary exposure to asbestos might increase the risk of cardiovascular disease, but, however, that further epidemiologic studies of asbestos workers did not support atherosclerosis being provoked by asbestos exposure. Dr. Federman opined that there was an increased risk of respiratory disease with pneumoconiosis, pleural cancer, and lung cancer being increased, as well as peritoneal cancer in the subgroup of asbestos workers, but no indication of increased risk of atherosclerotic cardiovascular disease or congestive heart failure within this group. He advised that the employee apparently had advanced atherosclerosis, congestive heart failure, and arrhythmias, which were the precipitating cause of his death, exacerbated by type 2 diabetes mellitus and cigarette exposure, as well as asbestos exposure. Dr. Federman reported that a medical study showed that former smokers had an elevated risk of ischemic heart disease and opined that asbestos was an independent contributor to cardiovascular disease, presumably from its inflammatory effect, but that smoking was a much more profound cause of inflammation. He concluded that there was no evidence of acceleration of his atherosclerosis from the occupational asbestos exposure.

The Board finds that the reports are of equal weight and as such are in conflict. The Board will remand the case for referral to an impartial medical specialist pursuant to 5 U.S.C. § 8123(a). After such further development as OWCP deems necessary, it should issue an appropriate decision.

CONCLUSION

The Board finds that there is an unresolved conflict in medical opinion regarding whether the employee's asbestosis caused or contributed to his death. As such, the case is not in posture for decision.

ORDER

IT IS HEREBY ORDERED THAT the March 1, 2016 decision of the Office of Workers' Compensation Programs is set aside, and the case is remanded for further development consistent with this decision of the Board.

Issued: March 2, 2017
Washington, DC

Christopher J. Godfrey, Chief Judge
Employees' Compensation Appeals Board

Patricia H. Fitzgerald, Deputy Chief Judge
Employees' Compensation Appeals Board

Alec J. Koromilas, Alternate Judge
Employees' Compensation Appeals Board